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CASE OF DOUBLE HYDRONEPHROSIS WITH DILATATION OF THE BLADDER AND URETERS DUE TO DISEASE OF THE PROSTATE GLAND.

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The following case is one of special interest, both because a uniform double hydronephrosis is a rare disease, and because it was produced by a condition which proper early treatment might have remedied:

Thomas H., a printer by trade, aged 58 years. Health good previous to the present disease, which began two years ago.

The first symptoms were difficulty in urinating, retention of urine, irritation of the bladder, with all the phenomena of bladder tenesmus. These symptoms were shortly followed by incontinence and constant dribbling of urine. The patient's clothing was with difficulty kept dry, and the urinous odor consequent upon this condition was a source of great annoyance.

There was no constitutional disturbance up to three months ago, when uræmic phenomena began to develop. The appetite, strength and nutrition were unimpaired.

He had been under the care of various physicians, who called his ailment by different names, and gave as many different forms of treatment. He was treated at various times by tonics (strychnia and quinine), by diuretics (buchu and Bethesda water), and more latterly by full doses of ergot, on the diagnosis of diabetes insipidus.

I was unable to find evidence that there was any large increase in the amount of urine, but the constant dribbling rendered an estimate somewhat difficult.

The appearance and low specific gravity probably led to the diagnosis of polyuria.

I first saw the case December 17, '82. He then had not been under medical treatment for some time, but had been taking some proprietary medicine.

At this date he was evidently suffering from *uraemia*, and upon inquiry, I learned that he had been having violent headache in the morning for three months.

Loss of appetite, weakness and emaciation had been progressive for five weeks. Nausea and vomiting began four weeks previous to my first visit.

December 17, 1882—weakness and emaciation extreme—entire loss of appetite, constant nausea, with frequent distressing vomiting.

The most distressing symptom, however, was great dyspnœa, apparently due to emphysema of the lungs. No cough and no other lung disease, No evidence of heart disease. *No dropsy*.

The dyspnœa had developed but one week before I first saw the case. The amount of urine passed was measured for several days, and making allowance for that which dribbled away, it was about two pints in twenty-four hours. The urine which I examined was offensive, of alkaline reaction; spec. gr. 1005, and contained no albumen.

My diagnosis, from the general symptoms, was *Bright's disease*, and the treatment ordered hot baths and pilocarpus, tonics, stimulants, and milk. No relief followed the treatment; the

baths and pilocarpus failed to produce sweating, and caused so much distress that they were abandoned.

Specimens of the urine were submitted to Dr. Schaeffer for microscopical examination, December 19, 1882 and December 23, 1882—and the following are his reports:

Analysis of Urine, Dec. 19th.
No. 2064.
Dr. Prentiss.
Mr. H's Case.

Color, very pale; turbid.
Reaction neutral.
Specific gravity 1.010. Quantity said to be normal.
Trace of albumen.
No sugar.

Microscopical Examination:
Triple phosphates in small amount.
Some altered blood.
No pus, casts or renal epithelium.
Granular bladder epithelium.

E. M. SCHÆFFER, M. D.

Analysis of Urine, Dec. 23.
No. 2074.
Dr. Prentiss.
Mr. H's Case.

Color, very pale, slightly turbid.
Reaction neutral.
Specific gravity 1.012.

No albumen.

Microscopical Examination:
Phosphate of lime crystals and granular debris.
No blood, pus, or casts.
No indications of bladder or kidney disease.

E. M. SCHÆFFER, M. D.

Up to December 19 the patient still continued up, and even went out to carry the sample of urine to Dr. Schæffer on that date.

But after this he continued to grow rapidly worse, the nausea and vomiting and the dyspnœa were more aggravated, the mind became dull, and constant muscular twitchings threatened convulsions.

This condition was treated by rectal injections of

GRAMS.

℞ potass. brom. 12.
Spts. æth. co. 25.
Mist. asafœt. ad 200.

M: Two tablespoonfuls every four hours.

Death occurred, without convulsions, December 27, 1882.

Autopsy by Dr. N. Acker, December 28, assisted by Dr. E. M. Schæffer, Mr. Gurley (medical student), and myself.

Body greatly emaciated, rigor mortis well marked. Only the cavities of the abdomen and pelvis examined. Liver, spleen, pancreas, intestines and suprarenal capsule healthy. When the intestines were removed, the bladder was brought in view, appearing as a whitish, fibrous-looking, irregularly nodulated tumor, reaching nearly to the umbilicus.

It was distended with urine of the same character as that examined during life, and was estimated to contain about one quart.

It was partly emptied through the urethra by making compression, and the bladder, kidneys and ureters were carefully removed for further examination. After maceration in alcohol the bladder measured 18 c. m. (7 inches) in length, and 10 c. m. (4 inches) transverse diameter; the walls were greatly thickened and rough, traversed by a coarse network of fleshy cords resembling the columnæ carneæ of the heart.

In many places in between these bands were pouch-like dilatations, which gave to the external surface of the bladder a nodulated appearance.

The pericystic cellular tissue was congested and hypertrophied.

There was no obstruction to the outlet of the ureters, and apparently no dilatation of the ureters near the bladder.

We come now to refer to the most important pathological condition of all. It is *the peculiar enlargement of the middle lobe of the prostate gland, which projects into the neck of the bladder in such a manner as to form a valvular obstruction to the escape of urine.*

Each ureter was 30 c. m. (12 inches) long, dilated very much alike, irregularly from the size of a pencil to that of the thumb, the largest size being at the upper part where they widened out into the pelvis of the kidney.

The kidneys were both dilated to a capacity of about half a pint, and contained urine to that amount.

The kidney structure proper was reduced to from 1 to 2 c. m. (⅓ to ⅔ inches) in thickness.

There are several points of interest in this case, which render it instructive and interesting.

1. The obstruction which caused the hydronephrosis was undoubtedly due to the peculiar enlargement of the middle lobe of the prostate gland, which resembled in shape the crista galli of the ethmoid bone, and covered the orifice of the urethra like a valve.

There had been no history of renal colic during life, nor was there any evidence of renal calculus found after death.

The introduction of the catheter had not been practiced at any time during the treatment, probably because the dribbling of urine was looked upon as an incontinence, rather than an evidence of retention.

The early use of the catheter by relieving distension of the bladder would undoubtedly have prevented the development of hydronephrosis.

The patient's life would have been prolonged, and have been made more comfortable, although it cannot be said that he would not have ultimately succombed to chronic cystitis, so commonly a termination in greatly enlarged prostate.

When I was called to the case, the uræmic symptoms completely masked all evidence of local disease, and even if the exact diagnosis had been made,it was too late to accomplish any good to the patient. Fatal mischief was already done.

2. Examination of the urine failed to throw any light upon the character of the disease.

The analysis was made by Dr. Schæffer, who is an experienced and skilled microscopist, yet his report states there were "no indications of bladder or kidney disease."

An examination of the analysis justifies this statement, when made from the standpoint of the urine alone. There was found no pus, blood nor casts; no albumen nor sugar; the neutral reaction and low specific gravity alone marked a departure from the normal; yet, *clinically*, the patient was undoubtedly dying of uræmia.

The character of the urine was such as might have been expected under the conditions presented.

There was no disease of the secreting structure of the kidneys as in the forms of Bright's disease, and consequently none of the changes in the urine found in that disease were observed.

It was simply normal urine altered by the circumstances of pressure and re-absorption.

3. There was no dropsy. Dropsy, as is well known, is a constant attendant upon the uræmia of Bright's disease.

I have noticed in several cases of fatal uræmia due to chronic cystitis and chronic pyelitis, in which the parenchyma of the kidneys was not involved, that dropsy did *not* occur.

In these cases there is no albumen in the urine except such as may be accounted for by the presence of pus or blood, and the amount of urine is usually not materially affected until exhaustion begins to make itself manifest.

4. Death from uræmia in cases of hydronephrosis is not common, for the reason that hydronephrosis is seldom double. It is usually confined to one kidney.

When it is unilateral, the other kidney takes on increased function, and the system continues to be depurated of its urea. The usual mode of death is either from exhaustion produced by the pressure of the tumor upon surrounding parts, or by the bursting of the sac into the peritoneum, causing rapid death by peritonitis.

It may not be without interest to recapitulate briefly the causes which lead to the development of hydronephrosis.

These may be divided into *extrinsic* and *intrinsic* causes.

Extrinsic, where a cause outside of the urinary apparatus is acting, such as the pressure of a tumor, cancer, uterine fibroid or ovarian tumor, or pregnancy; so also misplacements of the uterus may compress the ureter and produce backing up of the urine into the kidney.

Intrinsic, from a cause which acts within the urinary passages. The ob-

struction may be anywhere in the course of the urinary tract.

1. In the pelvis of the kidney, as of a calculus obstructing the orifice of the ureter.

2. In the ureter, usually a calculus.

3. In the bladder, and may be a calculus, or a tumor, or an abscess in the wall of the bladder, which obstructs the outlet of the ureter, or an enlarged prostate, as in the present case.

4. In the urethra,as an impacted calculus or a stricture; although such a cause of hydronephrosis is very rare.

As to the treatment of hydronephrosis, not much is to be said. It would depend upon the cause.

If extrinsic, such as misplacement of the uterus, it may be cured by relieving the misplacement. Usually the intrinsic causes are such that they are not amenable to treatment.

In enlarged prostate, the use of the catheter is imperative, and would prove curative of the hydronephrosis.

Cure is sometimes spontaneous by the escape of a calculus from the ureter into the bladder.

As a last resort, aspiration may be employed, or even nephrectomy practiced.